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Ludwig's angina

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The case is described of an occurrence of Ludwig's angina with advanced stage of the disease with progressive and rapid airway compromise and fatal consequence. A review of the literature is undertaken to gain a better understanding of the disease, and gives the opportunity for presenting a summary of the key issues regarding this dreaded disease, particularly the immediate management of it in the emergency department.

A previously healthy 40 year old man presented to our accident and emergency department with difficulty breathing. He had been taking antibiotics for a week for a sore throat that had developed after dental extraction. His symptoms had worsened with rapidly increasing shortness of breath during the past 24 hours. On arrival to our department, he had noisy (gurgling) breathing with drooling saliva, dyspnoea, dysphagia, and trismus. There was bilateral submandibular tense swellings and cellulitis, larger on the left. He was not able to protrude his tongue, which appeared elevated. He was feverish (39.2℃) and had a pulse rate of 130/min. His blood pressure was 180/94 and a respiratory rate of 25/min. His oxygen saturation was 95% on air and 99% on 15 l/min oxygen. Our immediate management of the patient included maintaining his upright sitting posture with continuous high oxygen flow administration.

The anaesthetic and ENT teams were involved immediately. He was given 6 mg of intravenous dexamethasone, 1.2 g of augmentin, intravenous hydration, and nebulised adrenalin. Twenty minutes from arrival, the patient's clinical picture had not changed. As he appeared stable, a decision was made for the immediate transfer of the patient to the operating theatre for an emergency tracheostomy under local anaesthetic followed by drainage of the submandibular swellings under general anaesthetic. In the operating room and before transferring the patient onto the operating table he developed an acute complete airway obstruction and respiratory arrest. An immediate tracheostomy and intubation performed. Asystole cardiac arrest followed. The patient was successfully resuscitated five minutes after arrest. The floor of the mouth was then drained. The patient was transferred to the intensive care unit. He unfortunately developed hypoxic ischaemic encephalopathy with failure to gain consciousness, decerebrate extensor spasms of the upper and lower limbs, and recurrent epileptic fits. He was eventually weaned off the ventilator and started breathing spontaneously. The neck swelling subsided and the drain was removed. He was then transferred to the neurological rehabilitation ward. He died of bilateral lower lobe bronchopneumonia, three weeks after his admission.

COMMENT

Ludwig's angina is best described by Karl Friedrich Wilhelm von Ludwig in 1836, as a rapidly and frequently fatal progressive gangrenous cellulitis and oedema of the soft tissues of the neck and floor of the mouth. It originates in the region of the submandibular gland with elevation and displacement of the tongue. The disease extends by continuity rather than lymphatic spread. Airway compromise has been recognised as the leading cause of death. Mortality exceeded 50%, but since the introduction of antibiotics in 1940s, improved oral and dental hygene, and aggressive surgical approach the mortality was reduced significantly. This resulted in the rare occurrence of the disease leaving many physicians with increasingly limited experience of Ludwig's angina.

Most Ludwig's angina infections are odontogenic.[1] Other causes include peritonsillar or parapharyngeal abscesses, mandibullar fracture, oral lacerations/piercing, or submandibullar sialodentitis. Predisposing factors include: dental carries, recent dental treatment, systemic illnesses such as diabetes mellitus, malnutrition, alcoholism, compromised immune system such as AIDS, and organ transplantation and trauma.[2–5] In children, it can occur de novo, without any apparent cause.[6 7] Early recognition of the disease is of paramount importance. Painful neck swelling, tooth pain, dysphagia, dyspnoea, fever, and malaise are the most common complaints. Neck swelling and a protruding or elevated tongue are seen in the vast majority. Stridor, trismus, cyanosis, and tongue displacement suggest an impending airway crisis. Oedema and induration of the anterior neck, often with cellulitis, may be present in advanced cases. Early signs and symptoms of obstruction may be subtle.

Airway compromise is always synonymous with the term Ludwig's angina, and it is the leading cause of death. Therefore, airway management is the primary therapeutic

concern.[8] The treatment plan for each patient should be individualised and based on a number of factors. The stage of the disease and comorbid conditions at the time of presentation, physician experience, available resources, and personnel are all crucial factors in the decision making.[9] Immediate involvement of the anaesthetic and otolaryngology team is crucial. If surgical procedure is necessary, then airway control becomes mandatory. Airway observation policy is appropriate in selected cases of lesser severity. It entails aggressive medical treatment and close observation, monitoring, and regular examination.[10] Flexible nasotracheal intubation requires skills and experience, if it is not feasible, cricothyrotomy and tracheostomy under local anaesthetic may be required, and this is occasionally performed in the emergency department in those with advanced stage of the disease. Tracheostomy and cricothyrotomy can in such cases be associated with difficulties and complications.[11] Endotracheal intubation is associated with high rate of failure with acute deterioration in respiratory status resulting in emergency ''slash'' tracheostomy. Elective awake tracheostomy is a safer and more logical method of airway management in patients with a fully developed Ludwig's angina.[12]

Observations from previous case studies have suggested that the use of intravenous dexamethazone and nebulised adrenaline often allow intubation to be carried out under more controlled conditions, often avoiding the need for tracheostomy or criccothyroidotomy. An initial dose of 10 mg of dexamethasone is followed by 4 mg every six hours for 48 hours. The dexamethasone reduces oedema and cellulitis, it provides the initial chemical decompression protecting the airway and allows improved antibiotic penetration into the area.[13 14] Nebulised adrenaline (1 ml of 1:1000 diluted to 5 ml of 0.9% saline) is believed to be safe and effective in reducing upper airway obstruction of differing aetiologies.[15] Causative bacteria are usually a mix of aerobes and anaerobes, including mouth organisms such as streptococci or staphylococci.[7 8 12] High doses of penicillin G, with metronidazole or clindamycin or co-amoxiclav, are all good initial agents. Aerobic Gram negative organisms are uncommon in deep neck abscesses and the use of gentamicin is not recommended in the initial management.[12] The patient must be maintained in a sitting posture, and should never be left unattended. Other complications such as descending necrotising mediastinitis usually occur through the retropharyngeal space (71%) and the carotid sheath (21%).[16] The use of contrast enhanced computed tomography is not essential to confirm the diagnosis of Ludwig's angina but it is used to assess the extent of the abscess in all cases of retropharyngeal extension.[12]

In conclusion, Ludwig's angina is a potentially lethal disease. We believe that many accident and emergency departments have limited experience with the disease because of its rare occurrence. Management must entail early diagnosis and the immediate aggressive medical approach by the emergency, anaesthetic, and otolaryngology teams, with securing and maintaining the airway being the primary goal in all patients presenting with Ludwig's angina.

Contributors

K Saifeldeen was involved in management of the patient in the accident and emergency department, carried out the literature search, and wrote the paper. R Evans was the consultant ENT surgeon responsible for the care of the patient and contributed to the writing of the paper.

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